DISTRIBUTION OF THE Rh FACTOR IN AMERICAN INDIANS

By KARL LANDSTEINER, M.D., ALEXANDER S. WIENER, M.D., and G. ALBIN MATSON, Ph.D.

(From the Laboratories of The Rockefeller Institute for Medical Research, and the Office of the Chief Medical Examiner of the City of New York, New York, and the Station Hospital, Fort Lewis, Washington)

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Among the numerous investigations on the racial distribution of the human individual blood properties A, B, and M, N, the most striking deviations from other races were found in American Indians, Eskimos, Australian aborigines, and Ainus.[1] Since material from American Indians was available to us, it was considered of interest to study this with regard to the incidence of the Rh factor.

Materials and Methods

The clotted blood samples obtained by venepuncture at Fort Lewis, Washington, were shipped at once by air mail to New York City, where they arrived in good condition. These specimens came from Indian patients at the Tacoma Hospital and Indian soldiers stationed at Fort Lewis.

The Indians from which the samples were obtained fall into ten different linguistic groups with the exception of a few individuals. (This classification is based on the ethnological charts of linguistic stocks in Wissler's "The American Indian," 1938 edition, supplemented by information obtained from Dr. J. H. Hendry, Superintendent of the Indian Hospital, and Mr. O. C. Upchurch, Indian Agent at Tulalip, Washington.) The principal tribes of the individuals tested belonged to the following stocks. Shahaptin, Salishan, Klamath, Athabascan, Siouan, Tlingit or Koluschan, Haida, Shoshonean, Eskimauan, Algonkin.

Each Indian tested was classified according to the "degree" of Indian blood. The ratings as to racial purity are based on the agency records; these, naturally, may not be entirely accurate.

When the blood samples were received, even suspensions were prepared by breaking up the clots in saline solution, removing coarse particles, and washing once with saline. The tests for Rh were made with guinea pig immune sera (11) and with a human anti-Rh serum. For the latter tests, equal parts of a thin blood suspension and of serum were incubated at 37°C. All specimens designated as derived from

[1] Illustrative results selected from the literature can be found in the papers by Snyder (1) (A, B in Indians), Matson and Schrader (2) (A, B in Indians), Landsteiner and Levine (3) (M, N in Indians), Birdsell and Boyd (4) (A, B, M, N in Australian aborigines), Fabricius-Hansen (5) (A, B, M, N in Eskimos), Grove (6) (A, B in Ainus and Filipinos), Nigg (7) (A, B in Polynesians and Hawaiians), Kubo (8) (M, N in Ainus); and for general bibliography see Boyd (9) and Wiener (10).

full blooded and three-quarter blooded Indians were tested with both reagents, the others with human serum alone. In addition, the blood groups, sub groups, and M-N types were determined.

Some of the specimens from full blooded and three-fourths blooded Indians were examined with a human serum[2] which in whites gives about 27 per cent negative reactions instead of 15 per cent (11, 12).

Findings

The results presented in Table I demonstrate that the distribution of the Rh factor among American Indians differs widely from that among white Americans in New York City. While as many as 15 per cent of bloods from white individuals lack the Rh factor, only one such blood was found among 120 full blooded Indians and, of course, it cannot be concluded with certainty that even this single case was not attributable to some untraceable crossing

TABLE I

Distribution of the Rh Agglutinogen among American Indians As Compared with White Individuals

Population examined	Rh-positive		Rh-negative		Total No. examined
	Number	Per cent	Number	Per cent	
Full blooded Indians	119	99.2	1	0.8	120
Whites (New York City)					
Landsteiner and Wiener (11)	379	84.6	69	15.4	448
Levine *et al.* (22)		86		14	1035

with whites, a possibility which may also apply to the single group B individual found (*cf.* Table II). Among 69 Indians designated as three-fourths blooded all gave positive reactions with the exception of one blood, which was negative with the guinea-pig antiserum and with one human serum, but gave weakly positive reactions with three other human anti-Rh sera.

In the tests for the type demonstrable with the special human anti-Rh serum mentioned above, 69 specimens from full blooded Indians were examined. Of these, 29, or about two-fifths, showed negative reactions with this serum which is more than twice the frequency found among Rh-positive bloods from white individuals.

In Table II are given the figures for the distributions of O-A-B, A_1-A_2, and M-N. These confirm previous findings, especially the high frequency of group O and the low incidence of type N. The incidence of group A is higher in our material than in most Indian tribes, but considerably lower than in two tribes studied by Matson and Schrader (2), and it should be noted that, as in a

[2] For this serum we are indebted to Dr. Philip Levine.

previous study by Levine, Matson, and Schrader (13), all the group A full blooded Indians belonged to subgroup A_1.

The frequencies of the various blood factors in Indians known not to be of pure stock are presented in Table III, together with the frequencies to be anticipated from the degree of mixture with whites.

TABLE II

Distribution of the Agglutinogens A_1-A_2-B, and M-N among American Indians As Compared with American Whites

Population		Distribution of groups and subgroups						Distribution of M-N types		
		O	A_1	A_2	B	A_1B	A_2B	M	N	MN
Full blooded Indians (Present study)	Number....	88	31	0	1	0	0	68	5	47
	Per cent....	73.3	25.8	0	0.8	0	0	56.7	4.2	39.2
American whites (Cited after Wiener (10))	Per cent....	44.6	25.6	12.5	13.6	3.1	1.2	29.2	21.3	49.6

TABLE III

Distribution of the Agglutinogens Rh, A_1-A_2-B, and M-N among American Indians of Mixed Ancestry

(Compared with expected distribution calculated from average degree of crossing with whites)

Population		Distribution of Rh		Distribution of blood groups						Distribution of M-N		
		Positive	Negative	O	A_1	A_2	B	A_1B	A_2B	M	N	MN
American Indians of mixed ancestry (Present study)	Number...	148	7	90	49	5	7	4	0	75	11	69
	Per cent...	95.5	4.5	58.1	31.6	3.2	4.5	2.6	0	48.4	7.1	44.5
Theoretical distribution for populations with 43 per cent Indian blood	Per cent...	93.5	6.5	56.1	25.9	7.4	8.4	1.6	0.5	40.4	13.2	46.3
60 per cent Indian blood	Per cent...	95.6	4.4	61.0	25.7	4.4	6.6	1.2	0.3	45.4	10.6	43.9

It may be recalled that the expected distribution resulting from interbreeding can easily be computed from the number of individuals of the parent populations and the distribution of the blood properties in each of them. This has been done for the blood groups by Bernstein (14), who showed that the distribution in the mixed population can be obtained graphically by a method analogous to the location of the center of gravity of two masses. In this way, the frequency of the *rh* gene for half blooded Indians would be the average

of the frequency of this gene in whites and full blooded Indians, respectively, namely, equal to

$$\tfrac{1}{2}\,(\sqrt{0.15} + \sqrt{0.008})$$

or 25 per cent, so that Rh-negatives should have the frequency $(0.25)^2$ or about 6 per cent (or about 4 per cent if the single Rh-negative individual among the full blooded Indians is discounted).

From the data on the ancestry of each individual, the average degree of crossing with whites was calculated for the 155 Indians of mixed stock. This proved to be almost 43 per cent Indian and 57 per cent white blood. The expected distribution of the blood properties in the series tested was calculated as indicated above. According to expectation, the observed frequencies were intermediate between those for whites and full blooded Indians but, as shown in Table III, the observed figures tally somewhat better with the computed distribution for a population with a higher proportion of Indian blood. The discrepancy may well be accidental considering the relatively small size of the series; in addition, the white population selected as standard may be different from that with which the crossing occurred, and finally the Agency records are subject to some error.

DISCUSSION

The variations in the distribution of individual blood properties among different races have given rise to several theories concerning their origin. Emphasis has been laid on the effects of interbreeding of races (migration), isolation of small groups, and mutation after the establishment of races (*cf.* Boyd (15), Gates (16), Wyman and Boyd (17)).

The case of the American Indians is of particular interest in the discussion of these problems. When practically no individuals with A and B agglutinogens were found at first, it was concluded that Indians originally all belonged to group O, and the occurrence of groups A and B was due to racial admixture. These ideas had to be abandoned when Matson *et al.* (2, 18) found that some tribes of Indians had 75 to 83 per cent of group A individuals (*cf.* Boyd (19)).

For the sponsors of the mutation theory the difficulty presented itself of accounting for the absence of agglutinogens A and B in the majority of Indian tribes, which would require that, for unknown reasons, in just those tribes, in contrast to other populations, no mutations from groups O to A or B had occurred, or at most, only exceptionally. In this regard the computation made by Wyman and Boyd (*v.* Haldane (20)), using a formula derived by Fisher (21), should be cited, from which it appears that the present distribution of the groups in various races throughout the world can only be explained by postulating an improbably high rate of mutation, if this took place after the differentiation of the present races.

As an explanation it would seem most reasonable, in agreement with Matson and Schrader, and Boyd, to attribute the unusual distribution of the blood properties in Indians, and the marked differences in frequencies of groups A and O among various tribes, to the geographic isolation of small numbers of individuals. However, it is not immediately clear why the examined Indian tribes differ with regard to groups A and O but all show the same unusual high frequency of type M and have no A_2 and B, and if our findings should be duplicated in investigations on other tribes of Indians, why all possess the factor Rh. One could think that there were successive separations of small groups, resulting by chance in segregation of tribes consisting mainly of group A or group O, but sharing the same original high incidence of genes for type M and Rh. A parallel case would be the wide variations in the incidence of the four blood groups among Eskimos in various localities, provided the very high incidence of type M (even higher than among Indians in the only tribe (5) tested for this property) is found to be a general attribute of this race.

SUMMARY

Erythrocytes from 120 full blooded American Indians and 155 Indians of mixed ancestry were tested for the Rh agglutinogen. Only a single blood among the full blooded Indians appeared to lack this factor, and in the Indians known not to be full blooded, the distribution of this (and other) blood properties was found to be intermediate between that for whites and pure Indians according to expectation. A variant of Rh demonstrable by a special human serum was more than twice as frequent in full blooded Indians as in white individuals.

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